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EXAMINER

LI, RUIXIANG

ART UNIT

PAPER NUMBER

1646

DATE MAILED: 06/13/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

**Office Action Summary**

Application No.

09/992,238

Applicant(s)

BATTAGLINO ET AL.

Examiner

Ruixiang Li

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-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

**Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

**Status**

- 1) ☒ Responsive to communication(s) filed on 15 October 2002.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

**Disposition of Claims**

- 4) ☒ Claim(s) 23-26, 28, 30, 31, 34-36, 40, 41 and 44-46 is/are pending in the application.
- 4a) Of the above claim(s) \_\_\_\_\_ is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 23-26, 28, 30, 31, 34-36, 40, 41 and 44-46 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

**Application Papers**

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- 11) ☐ The proposed drawing correction filed on \_\_\_\_\_ is: a) ☐ approved b) ☐ disapproved by the Examiner.  
If approved, corrected drawings are required in reply to this Office action.
- 12) ☐ The oath or declaration is objected to by the Examiner.

**Priority under 35 U.S.C. §§ 119 and 120**

- 13) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).  
a) ☐ All b) ☐ Some \* c) ☐ None of:  
1. ☐ Certified copies of the priority documents have been received.  
2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.  
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).  
\* See the attached detailed Office action for a list of the certified copies not received.
- 14) ☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).  
a) ☐ The translation of the foreign language provisional application has been received.
- 15) ☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

**Attachment(s)**

- 1) ☐ Notice of References Cited (PTO-892) 4) ☐ Interview Summary (PTO-413) Paper No(s). \_\_\_\_\_
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948) 5) ☐ Notice of Informal Patent Application (PTO-152)
- 3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) 12, 13. 6) ☐ Other: \_\_\_\_\_

## **DETAILED ACTION**

### **I. Status of Application, Amendments, and/or Claims**

The amendment filed in Paper No. 15 on April 4, 2003 has been entered in full. Claims 30 and 31 have been amended. Claims 27, 29, 32, 33, 37-39, 42-43, and 47-48 have been canceled. Claims 23-26, 28, 30, 31, 34-36, 40, 41, and 44-46 are pending and under consideration.

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office Action.

### **II. Miscellaneous**

The Examiner acknowledges a spelling error in the First Office Action: " NFTA" should be "NFAT".

### **III. 35 U.S.C. § 101**

The rejection of claims 23-26, 28, 30, 31, 34-36, 40, 41, and 44-46 under 35 U.S.C. §101, as set forth at pages 3-5 of the previous Office Action (Paper No. 11, December 4, 2002), remains.

At page 5 of the Response, Applicants argue that the claimed subject matter is specific in that it is directed to methods of identifying compounds that modulate a specific G-protein coupled receptor, HGPRBMY8, and not simply any G-protein coupled

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receptor and that the claims further specify that the compounds identified by such method be useful for the treatment of caudate nucleus disorders, and not simple any disorder. Applicants assert that the utility is particular to the claimed subject matter and is specific.

This has been fully considered but is not deemed to be persuasive for the following reasons. First, in contrast with Applicants' argument, only claim 44 has the limitation that the candidate compound is useful for treating disorders of the caudate nucleus. The specification simply asserts that the present invention provides methods for treatment or prevention of cancers, immune disorders, or neurological disorders involving administering to an individual in need of treatment or prevention an effective amount of a purified antagonist of the HGPRBMY8 polypeptide ([0026] of page 6). No specific disorders that can be treated by a modulating compound of the HGPRBMY8 polypeptide have been clearly identified. Likewise, the list of disorders recited in claim 44 includes a variety of neurological disorders with different pathological conditions. Thus, Applicants have not identified a particular disorder that can be treated by modulating compounds identified by the claimed method. Secondly, a method that simply uses a specific putative GPCR does not render the method a specific utility, as applicants have argued. In the instant case, since the specification fails to disclose biological functions or activities of the HGPRBMY8 polypeptide that can be used in the present method to identify a modulating compound of the putative GPCR, the claimed method does not have a specific utility.

At the bottom of page 5 to first paragraph of page 6; third paragraph of page 7; and middle of page 9 of the Response, Applicants argue that the compounds identified by the claimed method meet the substantial utility as they are useful for treating specific disorders, preferably caudate nucleus disorders. Applicants argue that the HGPRBMY8 polypeptide is predominately expressed in caudate nucleus tissue of the brain. In fact, HGPRBMY8 was expressed at levels 825 fold higher relative to other tissues within the brain.

This has been fully considered but is not deemed to be persuasive for the following reasons. First, the claimed invention is drawn to a method of screening for candidate compounds modulating the activity of HGPRBMY8 polypeptide (claim 44 is drawn to a method of screening for candidate compounds modulating the activity of HGPRBMY8 polypeptide, which are useful for treating disorders of the caudate nucleus). However, the instant disclosure fails to disclose the biological functions or activities of the HGPRBMY18 polypeptide to be used for the screening candidate compounds, and more importantly the implication of the HGPRBMY18 polypeptide/nucleic acid in the disorders listed in claim 44. The expression data in Figs. 7 and 8 merely show that HGPRBMY8 was highly expressed in brain, particularly in caudate nucleus (see Example 4). They do not show that there is a differential expression between normal brain tissues and diseased tissues; they do not show a causative link between the HGPRBMY18 nucleic acid or polypeptide and the pathogenesis of disorders of the caudate nucleus. The specification fails to disclose any

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evidence on whether any disorders of the caudate nucleus can be treated by either up regulating or down regulating the activity of the HGPRBMY8 polypeptide.

Secondly, the claims recite "candidate compounds modulating the activity of HGPRBMY8 polypeptide". In other words, the candidate compounds may inhibit the activity of HGPRBMY8 polypeptide (antagonists or inhibitors) or enhance the activity of HGPRBMY8 polypeptide (agonists or enhancers). However, the disclosure fails to disclose the specific disorders that are treated by each type of identified candidate compounds.

Thirdly, claim 44 recites "disorders of the caudate nucleus", which encompasses a variety of neurological disorders. However, the disclosure fails to disclose any causative link of the HGPRBMY8 polypeptide/nucleic acid to any of these disorders. Furthermore, the specification fails to disclose whether an agonist or an antagonist (inhibitor) of the HGPRBMY8 polypeptide may be used to treat each of the listed disorders. It is even unclear whether these disorders are linked to the aberrant HGPRBMY8 expression. In view of the complexity of the pathological conditions of the neurological disorders, as listed in claim 44, it would take significant further research to determine (i) the biological functions of the HGPRBMY8 polypeptide or its implication in a caudate nucleus disorder; (ii) the activity to be used in the method for screening candidate compounds; and (iii) whether a specific candidate compound is useful for the treatment of a specific caudate nucleus disorder. Such significant further research is not permitted under 35 USC § 101. See *Brenner v. Manson*, 383 U.S. 519, 148 USPQ 689 (Sup. Ct. 1966), noting that "a patent is not a hunting license. It is not a reward for the

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search, but compensation for its successful conclusion." Thus, the specification fails to provide a substantial utility.

At second paragraph of page 6, Applicants argue that (i) that G-protein coupled receptors represent 45% of all drug targets for present-day therapies and continue to represent a significant focal point for drug discovery; and (ii) that since the claimed invention related to methods of screening for compounds that modulate a particular GPCR, which are common pharmaceutical practice, these compounds have a specific and substantial utility, and representing a real-world context use.

Applicants' argument has been fully considered but is not deemed to be persuasive for the following reasons. That GPCRs are common drug targets does not support that the HGPRBMY8 polypeptide plays a significant role in disorders of caudate nucleus. Commercial success is not an indication of a patentable utility whereas the commercial value does not simply render the claimed invention a specific and substantial utility. This is because many products may be commercially successful due to reasons unrelated to the use of the products. For example, a pharmaceutical company may wish to purchase a putative GPCR on the chance that it may turn out to be a drug target in future, even though determining such possibility requires substantial further experimentation. However, such substantial further experiment is not acceptable for patentable utility. In addition, substantial further experiment may have already been done on some of the GPCRs mentioned in the Response and specific functions may have already been known. This is not the case here.

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At third paragraph of page 6 of the Response, Applicants argue that specific and substantial caudate nucleus disorder utilities asserted by applicants are credible. The Examiner notes that since the asserted utility for the method is not specific and substantial, the credibility has not been assessed.

At the bottom of page 6 and bottom of page 9 of the Response, Applicants argue that PTO personnel must treat as true a statement of fact made by Applicants in relation to an asserted utility, unless countervailing evidence can be provided that shows that one of the ordinary skill in the art would have a legitimate basis to doubt the credibility of such a statement. Applicants argue that no such countervailing evidence has been provided. This has been fully considered but is not deemed to be persuasive because the Examiner believes that the *prima facie* case of lack of utility has been established for the reasons set forth above and in the Office Action (Paper No. 11).

At second paragraph of page 7; top to middle of page 9; 2<sup>nd</sup> paragraph of page 10 of the Response, Applicants argue that the claimed caudate nucleus utilities do represent a "real world" context of use since the methods of screening G-protein coupled receptors to identify modulators of the same are common pharmaceutical practice, and the fact that caudate nucleus disorders are "real" disorders afflicting the "world" today. This has been fully considered but is not deemed to be persuasive because the specification fails to show (i) whether there is a differential expression of HGPRBMY18 between normal brain tissues and diseased tissues; (ii) whether there is a



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causative link between the HGPRBMY18 nucleic acid or polypeptide and the pathogenesis of disorders of the caudate nucleus; (iii) whether any disorders of the caudate nucleus can be treated by either up regulating or down regulating the activity of the HGPRBMY8 polypeptide; and (iv) the specific disorders that are treated by each type of identified candidate compounds (antagonists or agonists). The Examiner further notes that the ligand and specific biological functions or other physiological significance of the GPCRs used the screening methods mentioned by the applicants in the Response may have already been identified. It is not the case here.

Beginning at the bottom of page 7 of the Response, Applicants argue that identification of the HGPRBMY8 ligand is not required to identify modulators of HGPRBMY8, identification of a ligand specific to HGPRBMY 8 is also not required to identify the biological function of HGPRBMY8. Applicants further argue that the specification already associates the HGPRBMY8 polypeptide with the incidence of caudate nucleus disorders, and that identification of the ligand of the HGPRBMY8 polypeptide is not required.

This has been fully considered but is not deemed to be persuasive for the following reasons. First, Applicants are mischaracterizing the Examiner's position. While identification of the HGPRBMY8 ligand is not required to identify modulators and biological functions of the HGPRBMY8 polypeptide, a specifically defined biological function or activity is required to practice the currently claimed invention, because the claimed invention is drawn to a method of screening for candidate compounds

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modulating the activity of the HGPRBMY8 polypeptide. Clearly, without such an activity of the HGPRBMY8 polypeptide, an artisan would not be able to perform the claimed method. However, the specification fails to disclose any biological functions or activities of the HGPRBMY8 polypeptide that can be used in the method to identify modulating compounds. Secondly, Applicants' argument that the specification already associates the HGPRBMY8 polypeptide with the incidence of caudate nucleus disorders is simply not true. The specification merely discloses that HGPRBMY8 was highly expressed in brain, particularly in caudate nucleus; the specification does not show that there is a differential expression between normal brain tissues and diseased tissues; it does not show a causative link between the HGPRBMY8 nucleic acid or polypeptide and the pathogenesis of disorders of the caudate nucleus. Finally, even if the specification had disclosed an association of the HGPRBMY8 polypeptide with the incidence of caudate nucleus disorders, such an association would still not be able to be used in the claimed method for screening modulating compounds.

At the bottom of page 9 of the Response, Applicants argue that the subject HGPRBMY8 polypeptide is capable of constitutive coupling to G-proteins involved in a signal pathway known to be mediated by Gq/11 or G alpha 15/16 or Gs coupled receptors that inhibit NFAT response elements. Applicants argue that the subject HGPRBMY8 polypeptide is a G-protein coupled receptor.

This has been fully considered but is not deemed to be persuasive for the following reasons. First, the Examiner does not deny that the polypeptide set forth in

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SEQ ID NO: 2 is a putative cell surface GPCR that can act through increase in either cAMP or  $\text{Ca}^{2+}$  signal transduction pathways via alpha 15. However, the specification has not disclosed any specific biological functions of the polypeptide and thus the disclosure is insufficient to satisfy the utility requirement under 35 U.S.C. §101. The specification clearly states that the HGPRBMY8 polypeptide is identified as a candidate GPCR based upon sequence homology. Obviously, further research is required to identify its physiological functions. In addition, there is no single well-established utility for the GPCR family due to the great diversity in structures and functions of the GPCR family. The functions of each GPCR need to be determined individually.

At page 10, third paragraph of the Response, Applicants argue that *Brenner v. Manson* does not apply to the instant application. The Examiner notes that the cited case does apply to the instant application because the claimed invention lacks a specific and substantial utility.

Beginning at the bottom of page 10 of the Response, Applicants argue that modulators of HGPRBMY8 identified by the claimed method are useful for treating caudate nucleus disorders, such a utility is specific since it is directed to a single polypeptide; Such a utility is substantial since the screening methods to identify modulators are common pharmaceutical practice. This has been fully considered but is not deemed to be persuasive for the reasons set forth above.

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At page 11, third paragraph of the Response, Applicants argue that the claimed invention is credible. The Examiner notes that since the asserted utility for the method is not specific and substantial, the credibility of the utility has not been assessed.

Beginning at the middle of page 11 of the Response, Applicants argue that the claimed methods and the HGPRBMY8 polypeptide have a well-established utility. This has been fully considered but is not deemed to be persuasive for the following reasons. First, the specification clearly states that the HGPRBMY8 polypeptide is identified as a candidate GPCR based upon sequence homology and this putative cell surface GPCR acts through increase in either cAMP or  $\text{Ca}^{2+}$  signal transduction pathways via alpha 15. However, the specification fails to disclose any specific biological functions or activities of the HGPRBMY8 polypeptide that can be used in the method to identify modulating compounds. There is no single well-established utility for the GPCR family due to the great diversity in structures and functions of the GPCR family. The functions of each GPCR need to be determined individually. Thus, further research is required to identify its physiological functions. It is noted that the coupling of the putative GPCR with a G-protein is not indicative of a defined biological effect or activity. The evidence of record indicates that the present HGPRBMY8 polypeptide is an orphan GPCR: without defined ligand(s) and biological functions or any other physiological significance.

Secondly, Applicants' argument that novel association of the HGPRBMY8 polypeptide to caudate nucleus disorders is simply not true. The specification merely discloses that HGPRBMY8 was highly expressed in brain, particularly in caudate

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nucleus; the specification does not show that there is a differential expression between normal brain tissues and diseased tissues; it does not show a causative link between the HGPRBMY18 nucleic acid or polypeptide and the pathogenesis of disorders of the caudate nucleus. Finally, if the specification had disclosed an association of the HGPRBMY8 polypeptide with the incidence of caudate nucleus disorders, such an association would likely provide a patentable utility for the HGPRBMY8 polypeptide or nucleic acid. However, such an association would still not be able to be used in the claimed method for screening modulating compounds and thus would not provide a patentable utility for the claimed method.

#### **IV. Claim Rejections Under 35 U. S. C. § 112, 1<sup>st</sup> Paragraph**

(i) The rejection of claims 23-26, 28, 30, 31, 34-36, 40, 41, and 44-46 under 35 U.S.C. §112, 1<sup>st</sup> paragraph, as set forth at pages 5-7 of the previous Office Action (Paper No. 11, December 4, 2002), remains. Specifically, since the claimed invention is not supported by either a specific, substantial, and credible utility, or a well-established utility, one skilled in the art clearly would not know how to use the claimed invention. The basis for this rejection is set forth at pages 5-7 of the previous Office Action (Paper No. 11, December 4, 2002).

The applicants' arguments about the patentable utility of the claimed invention has been fully considered but is not deemed to be persuasive for reasons set forth above.

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(ii) Furthermore, the previous Office Action (Paper No. 11, December 4, 2002) states that even if the claimed method of screening candidate compounds were to have a patentable utility, the instant disclosure would not be found to be enabling for the full scope of the invention as claimed in claim 44. This scope enablement rejection still remains.

Beginning at the middle of page 12 to the middle of page 16 of the Response, Applicants, based upon the *In re Wangs* factors, argue that the pending claims are fully enabled and would not require undue experimentation for a skilled artisan to make and use the invention. Applicants' lengthy argument has been fully considered but is not deemed to be persuasive for the following reasons. First, the Examiner notes that the issue related to scope enablement rejection is limited to claim 44, as set forth in the previous Office Action (Paper No. 11, December 4, 2002). Claim 44 is drawn to a method of screening a candidate compound which is useful for treating disorders of the caudate nucleus.

Secondly, Applicants' argument base upon the *In re Wangs* factors totally ignores the fact that the specification fails to identify the biological functions of the claimed polypeptide, fails to demonstrate the existence of link established between the molecules of the present invention and disorders of the caudate nucleus, and fails to demonstrate the likelihood of the success of treating disorders of the caudate nucleus with a potential candidate compound. None of the working examples are related to how to use the claimed method to identify modulating compounds of the putative GPCR and how to use such modulating compounds to treat the wide range of neurological

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diseases. No sufficient guidance has been provided in the specification regarding how to use the claimed method to identify modulating compounds and how to use such modulating compounds identified to treat the neurological diseases. What is disclosed in the specification is the high expression in brain, particularly in caudate nucleus, of a partially characterized putative GPCR mainly or an orphan GPCR without defined ligand(s) and biological functions or any other physiological significance.

Furthermore, the list of disorders of caudate nucleus recited in claim 44 includes a variety of disorders with different pathological conditions, ranging from a neurological disorder, Parkinson's disease, anxiety, manic depression to neoplastic disease of the brain, just to name a few. Thus, the claim is extremely broad. The Examiner notes that while the relative skill of those in the art is high in recombinant DNA technology and method of screening technology, successful treatment of the neurological disorders such as those recited in the claim remains a challenge.

Finally, as amended, claims 30 and 31 recite a reference polynucleotide. It is noted that the specification only enables the specific reference polynucleotide disclosed in the specification (see [0268]), but does not enable any other reference polynucleotides.

Accordingly, the specification fails to enable the claimed method. It would require undue experimentation for one skilled in the art to make and use the claimed invention.

## **V. Claim Rejections Under 35 U. S. C. § 112, 2<sup>nd</sup> Paragraph**

The rejection of claims 23-26, 28, 30, 31, 34-36, 40, 41, and 44-46 under 35 U.S.C. § 112, second paragraph, as set forth at page 7 of the previous Office Action (Paper No. 15, April 4, 2003), remains.

Applicants argue that the steps of the method clearly state that the criterion for selecting a candidate compound would be those “test compounds that modulate the activity of the G-protein coupled receptor polypeptide”. Applicants further argue that the preamble of claim 23 merely states the purpose or intended use of the invention. The purpose is to find “compounds that modulate the activity of a G-protein coupled receptor”, which is the same goal that is met by the last stated step of claim 23. Since the method is to identify modulators of HGPRBMY8, identifying compounds that affect the activity of HGPRBMY8 is a sufficient enough criterion to select a candidate compound.

This has been fully considered but is not deemed to be persuasive because the steps recited in the method fail to indicate clearly what activity of a G-protein coupled receptor polypeptide is detected or measured, rendering the claim 23 and its dependent claims indefinite.

## **VI. Conclusion**

No claims are allowed.



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Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire **THREE MONTHS** from the mailing date of this action. In the event a first reply is filed within **TWO MONTHS** of the mailing date of this final action and the advisory action is not mailed until after the end of the **THREE-MONTH** shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than **SIX MONTHS** from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Ruixiang Li whose telephone number is (703) 306-0282. The examiner can normally be reached on Monday-Friday, 8:30 am-5:00 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Yvonne Eyler, can be reached on (703) 308-6564. The fax phone number for this Group is (703) 305-3014 or (703) 308-4242.

Communications via Internet e-mail regarding this application, other than those under 35 U.S.C. 132 or which otherwise require a signature, may be used by the applicant and should be addressed to [yvonne.eyler@uspto.gov].

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All Internet e-mail communications will be made of record in the application file. PTO employees do not engage in Internet communications where there exists a possibility that sensitive information could be identified or exchanged unless the record includes a properly signed express waiver of the confidentiality requirements of 35 U.S.C. 122. This is more clearly set forth in the Interim Internet Usage Policy published in the Official Gazette of the Patent and Trademark on February 25, 1997 at 1195 OG 89.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the Group receptionist whose telephone number is (703) 308-0196.

*Elizabeth C. Kemmerer*

Ruixiang Li  
Examiner  
June 5, 2003

ELIZABETH KEMMERER  
PRIMARY EXAMINER